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=> s TIMP-1
6 FILES SEARCHED...
L1 8586 TIMP-1

=> s l1 and fusion
L2 425 L1 AND FUSION

=> s l2 and albumin
L3 241 L2 AND ALBUMIN

=> s albumin fusion protein
L4 8360 ALBUMIN FUSION PROTEIN

=> s l4 and (tissue inhibitor metalloproteinase-1)
8 FILES SEARCHED...
L5 0 L4 AND (TISSUE INHIBITOR METALLOPROTEINASE-1)

=> s l1 and (TISSUE INHIBITOR METALLOPROTEINASE-1)
L6 49 L1 AND (TISSUE INHIBITOR METALLOPROTEINASE-1)

=> s l6 and l4
L7 0 L6 AND L4

=> e rosen, c/au
E1 2 ROSEN ZVI M/AU
E2 7 ROSEN ZVI MICHAL/AU
E3 0 --> ROSEN, C/AU
E4 1 ROSENABUM S/AU
E5 1 ROSENACKER A F/AU
E6 1 ROSENACKER ARTHUR F/AU
E7 2 ROSENADA CEPERO R/AU
E8 1 ROSENAGER L/AU
E9 1 ROSENAK B/AU

E10 49 ROSENAK B D/AU
E11 9 ROSENAK BERNARD D/AU
E12 19 ROSENAK D/AU

=> e haseltine, w/au
E1 1 HASELTINE WILLIAM G/AU
E2 1 HASELTINE WILLIAM GAGE/AU
E3 0 --> HASELTINE, W/AU
E4 5 HASELTON A/AU
E5 2 HASELTON AARON/AU
E6 1 HASELTON B J/AU
E7 6 HASELTON C J/AU
E8 13 HASELTON C L/AU
E9 3 HASELTON CAROLE J/AU
E10 2 HASELTON D J/AU
E11 5 HASELTON D R/AU
E12 4 HASELTON DEBRA R/AU

=> s e1
L8 1 "HASELTINE WILLIAM G"/AU

=> s e2
L9 1 "HASELTINE WILLIAM GAGE"/AU

=> d 18 ti abs ibib tot

L8 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Physicochemical properties of mono- and diisocyanates
AB Liquid d., viscosity, sp. heat, and vapor pressure data are reported for a new class of mono- and diisocyanates.
ACCESSION NUMBER: 1986:540063 HCAPLUS
DOCUMENT NUMBER: 105:140063
TITLE: Physicochemical properties of mono- and diisocyanates
AUTHOR(S): Achorn, Peter J.; **Haseltine, William G.**; Miller, J. K.
CORPORATE SOURCE: Chem. Res. Div., Am. Cyanamid Co., Stamford, CT, 06904-0060, USA
SOURCE: Journal of Chemical and Engineering Data (1986), 31(4), 385-7
CODEN: JCEAAX; ISSN: 0021-9568
DOCUMENT TYPE: Journal
LANGUAGE: English

=> d 19 ti abs ibib tot

L9 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2004 ACS on STN
TI High pressure carbon-13 nuclear magnetic resonance relaxation study of 2-n-butyl-3-n-hexylnaphthalene
AB Unavailable
ACCESSION NUMBER: 1981:559261 HCAPLUS
DOCUMENT NUMBER: 95:159261
TITLE: High pressure carbon-13 nuclear magnetic resonance relaxation study of 2-n-butyl-3-n-hexylnaphthalene
AUTHOR(S): **Haseltine, William Gage**
CORPORATE SOURCE: Pennsylvania State Univ., University Park, PA, USA
SOURCE: (1981) 195 pp. Avail.: Univ. Microfilms Int., Order No. 8112809
From: Diss. Abstr. Int. B 1981, 42(1), 230
DOCUMENT TYPE: Dissertation
LANGUAGE: English

=> d his

(FILE 'HOME' ENTERED AT 12:08:47 ON 01 JUL 2004)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, HCPLUS, JAPIO, JICST-EPLUS, CEN, CEABA-VTB' ENTERED AT 12:09:20 ON 01 JUL 2004

L1 8586 S TIMP-1
L2 425 S L1 AND FUSION
L3 241 S L2 AND ALBUMIN
L4 8360 S ALBUMIN FUSION PROTEIN
L5 0 S L4 AND (TISSUE INHIBITOR METALLOPROTEINASE-1)
L6 49 S L1 AND (TISSUE INHIBITOR METALLOPROTEINASE-1)
L7 0 S L6 AND L4
 E ROSEN, C/AU
 E HASELTINE, W/AU
L8 1 S E1
L9 1 S E2

=> d l2 ti abs ibib 1-15

L2 ANSWER 1 OF 425 MEDLINE on STN
TI Regulated shedding of PAR1 N-terminal exodomain from endothelial cells.
AB G protein-coupled receptors can trigger metalloproteinase-dependent shedding of proteins from the cell surface. We now report that G protein-coupled receptors can themselves undergo regulated metalloproteinase-dependent shedding. The N-terminal exodomain of protease-activated receptor-1 (PAR1), a G protein-coupled receptor for thrombin, displayed regulated shedding in endothelial cells, which normally express this receptor. Cleavage occurred at a site predicted to render the receptor unresponsive to thrombin. A chimeric protein in which the N-terminal exodomain of PAR1 was fused to an unrelated transmembrane segment was shed as efficiently as PAR1, shedding of both proteins was stimulated by phorbol ester and by a PAR1 agonist. TNFalpha protease inhibitor-2 (TAPI-2), phenanthroline, and tissue inhibitor of metalloproteinase-3 (TIMP-3) but not **TIMP-1** or -2 inhibited such shedding. These and other data suggest that the information that specifies PAR1 shedding resides within its N-terminal exodomain rather than its heptahelical segment, that activation of protein kinase C or of PAR1 itself can stimulate PAR1 shedding in trans, and that ADAM17/TACE or a metalloproteinase with similar properties mediates PAR1 shedding. Regulated shedding reduced the amount of cell surface PAR1 available for productive cleavage by thrombin by half or more, but thus far we have been unable to demonstrate an effect of PAR1 shedding on cellular responsiveness to thrombin. Nonetheless, regulated shedding of G protein-coupled receptors represents a new mechanism by which signaling by this important class of receptors might be modulated.

ACCESSION NUMBER: 2004209919 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14982936
TITLE: Regulated shedding of PAR1 N-terminal exodomain from endothelial cells.
AUTHOR: Ludeman Matthew J; Zheng Yao Wu; Ishii Kenji; Coughlin Shaun R
CORPORATE SOURCE: Cardiovascular Research Institute, University of California, San Francisco 94143-0130, USA.
CONTRACT NUMBER: HL44907 (NHLBI)
 HL59202 (NHLBI)
 HL65185 (NHLBI)
 HL65590 (NHLBI)
SOURCE: Journal of biological chemistry, (2004 Apr 30) 279 (18)
 18592-9.
 Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200406
ENTRY DATE: Entered STN: 20040427
Last Updated on STN: 20040611
Entered Medline: 20040610

L2 ANSWER 2 OF 425 MEDLINE on STN
TI Engagement of CD44 promotes Rac activation and CD44 cleavage during tumor cell migration.
AB CD44 is a major cell surface adhesion molecule for hyaluronan, a component of the extracellular matrix, and is implicated in tumor metastasis and invasion. We reported previously that hyaluronan oligosaccharides induce CD44 cleavage from tumor cells. Here we show that engagement of CD44 promotes CD44 cleavage and tumor cell migration, both of which were suppressed by a metalloproteinase inhibitor KB-R7785 and tissue inhibitor of metalloproteinases-1 (TIMP-1) but not by TIMP-2. We also present evidence that blockade of metalloproteinase-disintegrin ADAM10 (a disintegrin and metalloproteinase 10) by RNA interference suppresses CD44 cleavage induced by its ligation. Engagement of CD44 concurrently induced activation of the small GTPase Rac1 and led to drastic changes in cell morphology and actin cytoskeleton with redistribution of CD44 to newly generated membrane ruffling areas. A fluorescence resonance energy transfer approach to visualize GTP-bound Rac1 in living cells revealed the localization of the active Rac1 in the leading edge of the membrane ruffling areas upon ligation of CD44. Taken together, our results indicate that the cleavage of CD44 catalyzed by ADAM10 is augmented by the intracellular signaling elicited by engagement of CD44, through Rac-mediated cytoskeletal rearrangement, and suggest that CD44 cleavage contributes to the migration and invasion of tumor cells.

ACCESSION NUMBER: 2004053693 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14623895
TITLE: Engagement of CD44 promotes Rac activation and CD44 cleavage during tumor cell migration.
AUTHOR: Murai Toshiyuki; Miyazaki Yoshihiro; Nishinakamura Hitomi; Sugahara Kazuki N; Miyauchi Takayuki; Sako Yasushi; Yanagida Toshio; Miyasaka Masayuki
CORPORATE SOURCE: Laboratory of Molecular and Cellular Recognition, Osaka University Graduate School of Medicine, Suita 565-0871, Japan.. murai@orgctl.med.osaka-u.ac.jp
SOURCE: Journal of biological chemistry, (2004 Feb 6) 279 (6) 4541-50.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200404
ENTRY DATE: Entered STN: 20040203
Last Updated on STN: 20040402
Entered Medline: 20040401

L2 ANSWER 3 OF 425 MEDLINE on STN
TI Novel bi- and trifunctional inhibitors of tumor-associated proteolytic systems.
AB Serine proteases, cysteine proteases, and matrix metalloproteinases (MMPs) are involved in cancer cell invasion and metastasis. Recently, a recombinant bifunctional inhibitor (chCys-uPA19-31) directed against cysteine proteases and the urokinase-type plasminogen activator (uPA)/plasmin serine protease system was generated by introducing the uPA receptor (uPAR)-binding site of uPA into chicken cystatin (chCysWT). In the present study, we designed and recombinantly produced multifunctional inhibitors also targeting MMPs. The inhibitors comprise the N-terminal inhibitory domain of human TIMP-1 (tissue inhibitor of matrix metalloproteinase-1) or TIMP-3, fused to chCys-uPA19-31 or chCysWT. As demonstrated by various techniques, these fusion proteins

effectively interfere with all three targeted protease systems. In in vitro Matrigel invasion assays, the addition of recombinant inhibitors strongly reduced invasion of ovarian cancer cells (OV-MZ-6#8). Additionally, OV-MZ-6#8 cells were stably transfected with expression plasmids encoding the various inhibitors. Synthesis and secretion of the inhibitors was verified by a newly developed ELISA, which selectively detects the recombinant proteins. Invasive capacity of inhibitor-producing cells was significantly reduced compared to vector-transfected control cells. Thus, these novel, compact, and small-size inhibitors directed against up to three different tumor-associated proteolytic systems may represent promising agents for prevention of tumor cell migration and metastasis.

ACCESSION NUMBER: 2003416801 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12956425
TITLE: Novel bi- and trifunctional inhibitors of tumor-associated proteolytic systems.
AUTHOR: Krol Janna; Sato Sumito; Rettenberger Peter;
Assfalg-Machleidt Irmgard; Schmitt Manfred; Magdolen
Viktor; Magdolen Ulla
CORPORATE SOURCE: Klinische Forschergruppe der Frauenklinik der Technischen
Universitat Munchen, Klinikum rechts der Isar, D-81675
Munchen, Germany.
SOURCE: Biological chemistry, (2003 Jul) 384 (7) 1085-96.
Journal code: 9700112. ISSN: 1431-6730.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200404
ENTRY DATE: Entered STN: 20030906
Last Updated on STN: 20040410
Entered Medline: 20040409

L2 ANSWER 4 OF 425 MEDLINE on STN
TI An ELISA for the detection of **TIMP-1** based on recombinant single chain Fv **fusion** proteins.
AB BACKGROUND: Altered serum levels of **TIMP-1** are an indicator of various pathological states. To quantitate **TIMP-1** in biological samples, we have previously isolated **TIMP-1** specific single-chain Fv fragments (scFvs) using phage-display. In the work presented here, we have used these scFvs to establish a **TIMP-1** ELISA based exclusively on recombinant scFv **fusion** proteins. METHODS: Two distinct **TIMP-1** specific scFvs were used as the antigen binding components after being genetically fused to the N-termini of two different **fusion** protein constructs. One **fusion** protein, comprising a CL domain, serves as a coating reagent, while the second **fusion** protein with a modified form of bacterial alkaline phosphatase is used as a detection reagent. A double antibody sandwich-ELISA was then established and optimized. RESULTS: An ELISA for the quantitation of tissue inhibitor of metalloproteinase (**TIMP**)**-1**, based entirely on recombinant antibody fragments, was developed as an alternative to assays using polyclonal antisera or monoclonal antibodies. Its performance was shown to compare well with a conventional ELISA. Finally, **TIMP-1** concentrations in the sera of sixty healthy individuals were determined. CONCLUSION: The assay described here provides a standardized, reliable and readily available means of quantitation of **TIMP-1** in a large number of blood samples.

ACCESSION NUMBER: 2003390396 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12927684
TITLE: An ELISA for the detection of **TIMP-1** based on recombinant single chain Fv **fusion** proteins.

AUTHOR: Wozniak Gordana; Obermayr Eva; Jeras Matjaz; Knezevic Mio;
Ruker Florian
CORPORATE SOURCE: Institute of Applied Microbiology, BOKU-University of
Natural Resources and Applied Life Sciences, Muthgasse 18,
1190 Vienna, Austria.
SOURCE: Clinica chimica acta; international journal of clinical
chemistry, (2003 Sep) 335 (1-2) 49-57.
Journal code: 1302422. ISSN: 0009-8981.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: (EVALUATION STUDIES)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200404
ENTRY DATE: Entered STN: 20030821
Last Updated on STN: 20040420
Entered Medline: 20040419

L2 ANSWER 5 OF 425 MEDLINE on STN
TI A quantitative in vitro model of smooth muscle cell migration through the arterial wall using the human amniotic membrane.
AB OBJECTIVE: The development of intimal hyperplasia involves smooth muscle cell (SMC) migration into the intima and proliferation. Matrix metalloproteinases and their tissue inhibitors play important roles in this process. In this study, we describe a novel in vitro model for studying SMC migration through the vessel wall. METHODS AND RESULTS: Human aortic SMCs (hASMCs) labeled with ^{125}I -iododeoxyuridine or unlabeled were grown on the stromal aspect of the human amniotic membrane. Mechanical damage to endothelial cells grown on the basement membrane and addition of growth factors or platelets were characterized for their effect on SMC migration into the stroma both by histological methods and by measuring the radioactivity associated with the membrane after removal of noninvasive SMCs. To assess the reliability of the model, the cells were infected with a recombinant adenovirus encoding the tissue inhibitor of metalloproteinase-1 (**TIMP-1**). Addition of a platelet-derived growth factor gradient stimulated hASMC infiltration into the stroma. This effect was abolished with **TIMP-1**-transduced hASMC, confirming that **TIMP-1** overexpression blocks SMC invasion of the stroma. CONCLUSIONS: This in vitro model of SMC migration in the vessel wall provides an inexpensive, quantitative, and reliable tool to study the molecular and cellular mechanisms of intimal hyperplasia.

ACCESSION NUMBER: 2003280408 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12676801
TITLE: A quantitative in vitro model of smooth muscle cell migration through the arterial wall using the human amniotic membrane.
AUTHOR: Kallenbach Klaus; Fernandez Harold A; Seghezzi Graziano;
Baumann F Gregory; Patel Sundeep; Grossi Eugene A; Galloway
Aubrey C; Mignatti Paolo
CORPORATE SOURCE: Seymour Cohn Cardiovascular Surgery Research Laboratory,
New York University School of Medicine, New York, NY 10016,
USA.
CONTRACT NUMBER: CA80476-01 (NCI)
SOURCE: Arteriosclerosis, thrombosis, and vascular biology, (2003 Jun 1) 23 (6) 1008-13.
Journal code: 9505803. ISSN: 1524-4636.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200402
ENTRY DATE: Entered STN: 20030617
Last Updated on STN: 20040302

Entered Medline: 20040226

L2 ANSWER 6 OF 425 MEDLINE on STN
TI IL-10 signaling via IL-10E1 is dependent on tyrosine phosphorylation in the IL-10R alpha chain in human primary prostate cancer cell lines.
AB Interleukin 10 (IL-10) stimulates rapid nuclear translocation and binding of a 22 kDa protein, termed interleukin 10 enhancer 1 (IL-10E1), to a novel enhancer element (i.e. HTE-1) of the tissue inhibitor of metalloproteinase-1 (**TIMP-1**) gene to upregulate **TIMP-1** expression. IL-10E1 signaling involves tyrosine phosphorylation of the IL-10R JAK1 (Janus kinase) and TYK2 (tyrosine kinase) receptor kinases and tyrosine phosphorylation of two tyrosine moieties (Y57 and Y62) of a LIM domain of the IL-10E1 protein. In this paper, the studies showed that two tyrosine residues (Tyr(446) and Tyr(496)) located in the cytoplasmic domain of the IL-10R alpha chain were required for receptor function, and for phosphorylation and activation of IL-10E1. Immunoprecipitation studies revealed that 12 amino-acid peptides encompassing either of these two tyrosine residues in phosphorylated form coprecipitated IL-10E1 and blocked ligand-dependent IL-10E1 phosphorylation in a cell-free system. In contrast, peptides containing serine substitutions for Tyr(446) and Tyr(496), and tyrosine-phosphorylated peptides containing Tyr(230) or Tyr(252/259) did not prevent IL-10E1 activation or signaling. To confirm these observations *in vivo*, **fusion** protein constructs were made between a modified form of green fluorescent protein or GFP and the intact IL-10E1 protein (IL-10E1-MmGFP) and n-terminal peptides of the IL-10E1 protein (i.e. nt-nls-MmGFP and mutant sequences identified as nt-nls mC61-MmGFP and nt-nls mY57/mY62-MmGFP peptides). Confocal microscopy revealed that IL-10 triggered transport to the nucleus of IL-10E1-MmGFP, nt-nls-MmGFP, and nt-nls mC61-MmGFP by 10-30 min in HPCA-10a (human prostate cancer cells; derived from Gleason sum 10 tumor tissue) cells. IL-10 failed to induce nuclear translocation of the mY57/mY62-MmGFP peptides with point mutations of the two tyrosine groups. Coinjection of nt-nls-MmGFP with the IL-10R Tyr(446) and Tyr(496) amino-acid residues completely blocked ligand signaling. Coinjection of peptides containing either serine substitutions for Tyr(446) and Tyr(496) or Tyr(230) and Tyr(252/259) failed to block nt-nls-MmGFP signaling. The data demonstrate that IL-10E1 is directly recruited to the ligand-activated IL-10R by binding to specific phosphotyrosine groups which control tyrosine phosphorylation of the LIM domain of the IL-10E1 protein (i.e. Y57/Y62 groups) and IL-10E1 activation.

ACCESSION NUMBER: 2003275748 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12802285
TITLE: IL-10 signaling via IL-10E1 is dependent on tyrosine phosphorylation in the IL-10R alpha chain in human primary prostate cancer cell lines.
AUTHOR: Stearns Mark E; Hu Youji; Wang Min
CORPORATE SOURCE: Department of Pathology and Laboratory, Drexel University College of Medicine, 15th and Vine Sts, Philadelphia, PA 19102-1192, USA.. Stearnsm1@aol.com
SOURCE: Oncogene, (2003 Jun 12) 22 (24) 3781-91.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200307
ENTRY DATE: Entered STN: 20030613
Last Updated on STN: 20030703
Entered Medline: 20030702

L2 ANSWER 7 OF 425 MEDLINE on STN
TI Tissue inhibitor of metalloproteinase 1 inhibits excitotoxic cell death in neurons.

AB The upregulation of **TIMP-1** following an excitotoxic injury has recently been hypothesized to be part of a general neuronal response that mediates long-lasting changes involved in tissue reorganization and possibly neuroprotection. In this study we have shown for the first time that within hours of applying **TIMP-1** in recombinant form or by adenovirus-mediated gene transfer, neurons are highly protected against excitotoxic injury. Neither **TIMP-3** nor a nonsecretable form of **TIMP-1** protected neurons. **TIMP-1** conferred highly significant protection to hippocampal cells exposed to a wide range of glutamic acid concentrations in both dissociated and organotypic hippocampal cultures. **TIMP-1** did not prevent apoptotic cell death or death mediated by chemical ischemia. The observed neuroprotection may be explained by a decrease in calcium influx into neurons following stimulation with glutamate. These findings have a fundamental implication for our understanding of the physiological role of secreted **TIMP-1** in the central nervous system.

ACCESSION NUMBER: 2003084319 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12595242
TITLE: Tissue inhibitor of metalloproteinase 1 inhibits excitotoxic cell death in neurons.
AUTHOR: Tan Hiang Khoon; Heywood Darren; Ralph G Scott; Bienemann Alison; Baker Andrew H; Uney James B
CORPORATE SOURCE: University Research Centre for Neuroendocrinology and MRC Centre for Synaptic Plasticity, University of Bristol, Marlborough Street, Bristol BS2 8HW, UK.
SOURCE: Molecular and cellular neurosciences, (2003 Jan) 22 (1) 98-106.
Journal code: 9100095. ISSN: 1044-7431.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 20030222
Last Updated on STN: 20030422
Entered Medline: 20030421

L2 ANSWER 8 OF 425 MEDLINE on STN
TI EMMPRIN-mediated MMP regulation in tumor and endothelial cells.
AB Tumor invasion and metastasis are multistep processes which require extracellular matrix remodeling by proteolytic enzymes such as matrix metalloproteinases (MMPs). The production of these enzymes is stimulated by many soluble or cell-bound factors. Among these factors, extracellular matrix metalloproteinase inducer (EMMPRIN) is known to increase in vitro stromal cell production of MMP-1, MMP-2 and MMP-3. In this study, we demonstrated that EMMPRIN-transfected MDA-MB-436 tumor cells displayed a more invasive capacity than vector-transfected cells in a modified Boyden chamber invasion assay. Using gelatin zymography and protein analyses, we showed that EMMPRIN-transfected cancer cells produced significantly more latent and active MMP-2 and MMP-3 than vector-transfected cancer cells. We found that EMMPRIN did not regulate MMP-1, MMP-9, membrane type-1 MMP (MT1-MMP) expression and had also no effect on the production of the specific tissue inhibitors of MMPs (TIMPs), **TIMP-1** and **TIMP-2**. We also demonstrated that tumor-derived EMMPRIN stimulated MMP-1, -2, and -3 without modification of MMP-9, MT1-MMP, **TIMP-1** and **TIMP-2** production in human umbilical vein endothelial cells (HUVEC). These data provide support for the role of EMMPRIN in tumor invasion, metastasis, and neoangiogenesis by stimulating extracellular matrix remodeling around tumor cell clusters, stroma, and blood vessels.

ACCESSION NUMBER: 2003043627 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12553375
TITLE: EMMPRIN-mediated MMP regulation in tumor and endothelial cells.

AUTHOR: Caudroy Stephanie; Polette Myriam; Nawrocki-Raby Beatrice;
Cao Jian; Toole Bryan P; Zucker Stanley; Birembaut Philippe
CORPORATE SOURCE: Inserm U514, IFR53, CHU Maison-Blanche, Reims, France.
CONTRACT NUMBER: R01-CA79866 (NCI)
SOURCE: Clinical & experimental metastasis, (2002) 19 (8) 697-702.
JOURNAL code: 8409970. ISSN: 0262-0898.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 20030130
Last Updated on STN: 20030214
Entered Medline: 20030212

L2 ANSWER 9 OF 425 MEDLINE on STN
TI Expression of **TIMP-1** in *Pichia pastoris*. Selection of
an anti-**TIMP-1** specific single-chain Fv antibody from
a large non-immune library.
AB To quantitate tissue inhibitor of metalloproteinase (**TIMP**)-
1 in biological samples, a strategy for isolation of monoclonal
antibodies was applied that employs a phage-displayed single-chain Fv
(scFv). In order to obtain sufficient amounts of **TIMP-1**
to use as an antigen, high-level expression in *Pichia pastoris* was
achieved under the control of the AOX-1 promotor. Purified protein
antigen was then used for panning to achieve enrichment of specific phage
from naive scFv library. In five subsequent panning rounds, antibody
fragments that display specificity to **TIMP-1** were
selected. Regions encoding scFv were subcloned into a vector allowing
production of scFv-alkaline phosphatase (AP) fusion proteins.
Two such conjugates displaying dose-dependent reactivity with **TIMP**
-1 were isolated and characterised, providing the basis for the
construction of a **TIMP-1** quantitation assay based
entirely on recombinant proteins.

ACCESSION NUMBER: 2002720496 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12482633
TITLE: Expression of **TIMP-1** in *Pichia*
pastoris. Selection of an anti-**TIMP-1**
specific single-chain Fv antibody from a large non-immune
library.
AUTHOR: Wozniak Gordana; Obermayr Eva; Jeras Matjaz; Knezevic Mio;
Ruker Florian
CORPORATE SOURCE: Institute of Applied Microbiology, University of
Agricultural Sciences, Muthgasse 18, 1190, Vienna, Austria.
SOURCE: Clinica chimica acta; international journal of clinical
chemistry, (2003 Jan) 327 (1-2) 171-9.
Journal code: 1302422. ISSN: 0009-8981.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200311
ENTRY DATE: Entered STN: 20021218
Last Updated on STN: 20031107
Entered Medline: 20031106

L2 ANSWER 10 OF 425 MEDLINE on STN
TI Gene transfer of the urokinase-type plasminogen activator
receptor-targeted matrix metalloproteinase inhibitor **TIMP-**
1.ATF suppresses neointima formation more efficiently than tissue
inhibitor of metalloproteinase-1.
AB Proteases of the plasminogen activator (PA) and matrix metalloproteinase
(MMP) system play an important role in smooth muscle cell (SMC) migration
and neointima formation after vascular injury. Inhibition of either PAs

or MMPs has previously been shown to result in decreased neointima formation in vivo. To inhibit both protease systems simultaneously, a novel hybrid protein, **TIMP-1.ATF**, was constructed consisting of the tissue inhibitor of metalloproteinase-1 (**TIMP-1**) domain, as MMP inhibitor, linked to the receptor-binding amino terminal fragment (ATF) of urokinase. By binding to the u-PA receptor this protein will not only anchor the **TIMP-1** moiety directly to the cell surface, it will also prevent the local activation of plasminogen by blocking the binding of urokinase-type plasminogen activator (u-PA) to its receptor. Adenoviral expression of **TIMP-1.ATF** was used to inhibit SMC migration and neointima formation in human saphenous vein segments in vitro. SMC migration was inhibited by 65% in Ad.**TIMP-1.ATF**-infected cells. Infection with adenoviral vectors encoding the individual domains, Ad.**TIMP-1** and Ad.**ATF**, reduced migration by 32% and 52%, respectively. Neointima formation in saphenous vein organ cultures infected with Ad.**TIMP-1.ATF** was inhibited by 72% compared with 42% reduction after Ad.**TIMP-1** infection and 34% after Ad.**ATF** infection. These data show that binding of **TIMP-1.ATF** hybrid protein to the u-PA receptor at the cell surface strongly enhances the inhibitory effect of **TIMP-1** on neointima formation in human saphenous vein cultures.

ACCESSION NUMBER: 2002673180 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12433840
TITLE: Gene transfer of the urokinase-type plasminogen activator receptor-targeted matrix metalloproteinase inhibitor **TIMP-1.ATF** suppresses neointima formation more efficiently than tissue inhibitor of metalloproteinase-1.
COMMENT: Comment in: Circ Res. 2002 Nov 15;91(10):861-2. PubMed ID: 12433829
AUTHOR: Lamfers M L M; Grimbergen J M; Aalders M C; Havenga M J; de Vries M R; Huisman L G M; van Hinsbergh V W M; Quax P H A
CORPORATE SOURCE: Gaubius Laboratory TNO-PG, Leiden, The Netherlands.
SOURCE: Circulation research, (2002 Nov 15) 91 (10) 945-52.
Journal code: 0047103. ISSN: 1524-4571.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 20021116
Last Updated on STN: 20030202
Entered Medline: 20021205

L2 ANSWER 11 OF 425 MEDLINE on STN
TI Elevated levels of soluble CD163 in sera and fluids from rheumatoid arthritis patients and inhibition of the shedding of CD163 by TIMP-3.
AB The aim of the present study was to evaluate levels of soluble CD 163 in sera and fluids from rheumatoid arthritis (RA) patients and elucidate the mechanism that regulates the shedding of CD163. Levels of soluble CD163 in sera and fluids from RA patients were examined by a sandwich enzyme immunoassay and Western blotting. To determine the effects of tissue inhibitors of metalloproteinase (TIMPs) on the shedding of CD163 from monocytes/macrophages, levels of soluble CD163 in cultures of monocytes/macrophages and the expression of CD163 on monocytes/macrophages in the presence or absence of TIMPs were examined by a sandwich enzyme immunoassay and flow cytometry, respectively. The clinical marker that was most associated with serum levels of soluble CD163 was levels of CRP. TIMP-3, but not **TIMP-1** or **TIMP-2**, inhibited the shedding of CD163 from monocytes/macrophages. It was shown that serum levels of soluble CD163 are a sensitive and reliable marker to monitor activated macrophages in synovitis from RA patients and the results imply that the responsible proteinase for the shedding of CD163 is not a member

of the matrix metalloproteinases, but is likely to be a member of ADAMs.

ACCESSION NUMBER: 2002499282 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12296867
TITLE: Elevated levels of soluble CD163 in sera and fluids from rheumatoid arthritis patients and inhibition of the shedding of CD163 by TIMP-3.
AUTHOR: Matsushita N; Kashiwagi M; Wait R; Nagayoshi R; Nakamura M;
Matsuda T; Hogger P; Guyre P M; Nagase H; Matsuyama T
CORPORATE SOURCE: Department of Immunology and Medical Zoology, School of Medicine, Kagoshima University, Kagoshima City, Japan.
SOURCE: Clinical and experimental immunology, (2002 Oct) 130 (1) 156-61.
Journal code: 0057202. ISSN: 0009-9104.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200211
ENTRY DATE: Entered STN: 20021004
Last Updated on STN: 20021213
Entered Medline: 20021113

L2 ANSWER 12 OF 425 MEDLINE on STN
TI Anti-tumour necrosis factor (TNF)-alpha therapy (etanercept) down-regulates serum matrix metalloproteinase (MMP)-3 and MMP-1 in rheumatoid arthritis.
AB OBJECTIVES: Matrix metalloproteinases (MMPs) are cytokine-modulated enzymes that play an important role in the pathogenesis of rheumatoid arthritis (RA) by inducing bone resorption and cartilage destruction. This study evaluated the modulation of serum and synovial MMPs and their inhibitor, tissue inhibitor of matrix metalloproteinases (TIMP)-1, by therapy with soluble tumour necrosis factor (TNF) alpha receptor (etanercept). METHODS: Serum samples were collected from 60 RA patients at baseline and after 8 or 12 weeks of treatment. Paired synovial biopsies were obtained from 11 patients at two time points, before and after 8 weeks of treatment. We measured serum levels of MMP-1, MMP-3 and TIMP-1 by ELISA. Immunohistological analysis of synovial tissue was performed using monoclonal antibodies specific for MMP-1, MMP-3 and TIMP-1. RESULTS: Etanercept therapy significantly down-regulated serum levels of MMP-3 and MMP-1 in parallel with the reduction in inflammatory parameters (C-reactive protein concentration and erythrocyte sedimentation rate) in RA patients. Baseline pretreatment serum levels of MMP-3 correlated with changes in clinical disease activity during therapy. No consistent changes in serum level of TIMP-1 were observed, while ratios of MMP-1 and MMP-3 to TIMP-1 were down-regulated following etanercept treatment. Immunohistochemical analyses revealed great interindividual variability, with generally a high level of expression of MMP and low expression of TIMP. No significant change in the pattern or number of positive cells occurred during therapy. CONCLUSIONS: In RA patients, etanercept therapy down-regulates serum levels of MMP-3 and MMP-1 and the ratio between MMPs and TIMP-1. This may be an important mechanism for the prevention of future development of joint damage.

ACCESSION NUMBER: 2002272699 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12011369
TITLE: Anti-tumour necrosis factor (TNF)-alpha therapy (etanercept) down-regulates serum matrix metalloproteinase (MMP)-3 and MMP-1 in rheumatoid arthritis.
AUTHOR: Catrina A I; Lampa J; Ernestam S; af Klint E; Bratt J; Klareskog L; Ulfgren A-K
CORPORATE SOURCE: Department of Rheumatology, Karolinska Hospital, Stockholm, Sweden.
SOURCE: Rheumatology (Oxford, England), (2002 May) 41 (5) 484-9.

PUB. COUNTRY: Journal code: 100883501. ISSN: 1462-0324.
England: United Kingdom
(CLINICAL TRIAL)
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 20020516
Last Updated on STN: 20030304
Entered Medline: 20020703

L2 ANSWER 13 OF 425 MEDLINE on STN
TI Identification of the active site of gelatinase B as the structural element sufficient for converting a protein to a metalloprotease.
AB Gelatinase B is a member of the matrix metalloproteinase family that efficiently cleaves gelatin, elastin, and types V and X collagen. To understand the contribution of the active site of the enzyme (amino acid residues 373-456) in these activities, we studied catalytic properties of a **fusion** protein consisting of maltose binding protein and the active site region of gelatinase B. We found that addition of the active site of gelatinase B, which corresponds to 12% of the total protein molecule, to maltose binding protein is sufficient to endow the protein with the ability to cleave the peptide substrates Mca-PLGL(Dpa)AR-NH(2) and DNP-PLGLWA-(D)-R-NH(2). The **fusion** protein hydrolyzed the Mca-PLGL(Dpa)AR-NH(2) peptide with the same efficiency as that of the stromelysin, k_{cat}/K_m approximately 1.07×10^{16} M(-)(1) h(-)(1). The **fusion** protein, however, was not able to degrade the large substrate, gelatin. Inhibition of the activity of the protein by EDTA suggested that its activity was metal dependent. ESR analyses indicated that the **fusion** protein bound one molecule of Zn(2+). In addition, Z-Pro-Leu-Gly-hydroxamate and **TIMP-1** inhibited the activity of the protein, suggesting that the structure of the active site of the **fusion** protein is similar to that of the other metalloproteinases. These data provide fundamental information about the structural elements required for transforming a protein to a metalloprotease.

ACCESSION NUMBER: 2002207044 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11939773
TITLE: Identification of the active site of gelatinase B as the structural element sufficient for converting a protein to a metalloprotease.
AUTHOR: Kaur Kuljeet; Zhu Kijeen; Whittemore Marilyn S; Petersen Richard L; Lichte Andrea; Tschesche Harald; Pourmotabbed Tayebeh
CORPORATE SOURCE: Department of Biochemistry, University of Tennessee Health Science Center, Memphis, Tennessee 38163, USA.
CONTRACT NUMBER: AR41843 (NIAMS)
SOURCE: Biochemistry, (2002 Apr 16) 41 (15) 4789-97.
Journal code: 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200205
ENTRY DATE: Entered STN: 20020410
Last Updated on STN: 20021218
Entered Medline: 20020517

L2 ANSWER 14 OF 425 MEDLINE on STN
TI Regulation of galectin-9 expression and release in Jurkat T cell line cells.
AB Ecalectin/galectin-9 was recently described as a novel eosinophil chemoattractant highly expressed in immune tissues. We investigated the regulation of galectin-9 expression and release in Jurkat (a T cell line)

cells. We demonstrated that medium and long-sized galectin-9 isoforms were constitutively expressed, and phorbol 12-myriastate 13-acetate (PMA) upregulated the level of galectin-9 mRNA in Jurkat cells. Western blotting and flow cytometry analyses revealed that PMA stimulation resulted in the upregulation of both intracellular and surface galectin-9 protein. The stimulated Jurkat cells simultaneously released evident eosinophil chemoattractant activity (ECA). Main ECA was adsorbed by both lactose and anti-galectin-9 antibody affinity column, suggesting that the ECA was ascribed to galectin-9. When Jurkat cells were stimulated with PMA in the presence of a BB94, a matrix metalloproteinase (MMP) inhibitor, but not tissue inhibitor of metalloproteinase-1 (**TIMP-1**), the release of galectin-9 was suppressed in a dose-dependent manner. We further found that calphostin c, a protein kinase c (PKC) inhibitor, weakly but significantly suppressed the release of galectin-9. The present data suggested that galectin-9 production in Jurkat cells is provoked by the stimulation with PMA and that some MMP and PKC is, at least, partly involved in the release of galectin-9 from Jurkat cells.

ACCESSION NUMBER: 2002154634 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11886844
TITLE: Regulation of galectin-9 expression and release in Jurkat T cell line cells.
AUTHOR: Chabot Sophie; Kashio Yumiko; Seki Masako; Shirato Yukako; Nakamura Kazuhiro; Nishi Nozomu; Nakamura Takanori; Matsumoto Ryoji; Hirashima Mitsuomi
CORPORATE SOURCE: Department of Immunology and Immunopathology, Kagawa Medical University, 1750-1 Ikenobe, Miki-Cho, Kita-gun, Kagawa 761-0793, Japan.
SOURCE: Glycobiology, (2002 Feb) 12 (2) 111-8.
Journal code: 9104124. ISSN: 0959-6658.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 20020312
Last Updated on STN: 20020907
Entered Medline: 20020906

L2 ANSWER 15 OF 425 MEDLINE on STN
TI Change in gene expression subsequent to induction of Pnn/DRS/memA: increase in p21(cip1/waf1).
AB Pnn (PNN) is a nuclear and cell adhesion-related protein. Previous work has suggested that Pnn/DRS/memA is a potential tumor suppressor involved in the regulation of cell adhesion and cell migration. Using the ecdysone-inducible mammalian expression system, a stable inducible GFP-tagged human Pnn gene (PNNGFP) expressing 293 cell line was created (EcR293-PNNGFP). Cells induced to express PNNGFP not only exhibited increased cell-cell adhesion but also exhibited changes in cell growth and cell cycle progression. cDNA array analyses, together with real time PCR, revealed that the effects of exogenously expressed Pnn on cellular behavior may be linked to the regulation of the expression of specific subset genes. This subset includes cell cycle-related genes such as p21(cip1/waf1), CDK4, CPR2; cell migration and invasion regulatory genes such as RhoA, CDK5, **TIMP-1**, MMP-7, and EMMPRIN; and MIC-1. Concordant with previous observations of Pnn-induced phenotype changes, genes coding for epithelial associated processes and cell division controls were elevated, while those coding for increased cell motility and cellular reorganizations were downregulated. We utilized p21 promoter-luciferase reporter constructs and demonstrated that a marked stimulation of p21 promoter activity in 293 cells correlated with increased Pnn expression. Taken together, these data indicate that Pnn may participate in the regulation of gene expression, thereby, positively promoting cell-cell adhesion, and negatively affecting cell migration and cell proliferation.

ACCESSION NUMBER: 2001444706 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11494129
TITLE: Change in gene expression subsequent to induction of Pnn/DRS/memA: increase in p21(cip1/waf1).
AUTHOR: Shi Y; Simmons M N; Seki T; Oh S P; Sugrue S P
CORPORATE SOURCE: Department of Anatomy and Cell Biology, University of Florida College of Medicine, Archer Road, Gainesville, Florida, FL 32610-0235, USA.
CONTRACT NUMBER: EY07883 (NEI)
SOURCE: Oncogene, (2001 Jul 5) 20 (30) 4007-18.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010813
Last Updated on STN: 20010903
Entered Medline: 20010830

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Derwent World Patents Index
IBM Technical Disclosure Bulletins

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DB=USPT; PLUR=YES; OP=OR

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<u>L6</u>	L5 and l4	3	<u>L6</u>
<u>L5</u>	rosen.in.	2229	<u>L5</u>
<u>L4</u>	TIMP-1 and L3	104	<u>L4</u>
<u>L3</u>	l1 and L2	40558	<u>L3</u>
<u>L2</u>	TIMP or tissue inhibitor metalloproteinase	266364	<u>L2</u>
<u>L1</u>	albumin fusion?	55072	<u>L1</u>

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Search Results - Record(s) 1 through 3 of 3 returned.

1. Document ID: US 6544761 B2

L6: Entry 1 of 3

File: USPT

Apr 8, 2003

US-PAT-NO: 6544761

DOCUMENT-IDENTIFIER: US 6544761 B2

TITLE: Human tissue inhibitor of metalloproteinase-4

DATE-ISSUED: April 8, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Greene; John M.	Gaithersburg	MD		
Rosen; Craig A.	Laytonsville	MD		

US-CL-CURRENT: 435/69.2; 514/12, 530/350, 536/23.5

Full	Title	Citation	Front	Review	Classification	Date	Reference	Abstract	Abstract	Claims	KWIC	Drawn De
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2. Document ID: US 6448042 B1

L6: Entry 2 of 3

File: USPT

Sep 10, 2002

US-PAT-NO: 6448042

DOCUMENT-IDENTIFIER: US 6448042 B1

TITLE: Human tissue inhibitor of metalloproteinase-4

DATE-ISSUED: September 10, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Greene; John M.	Gaithersburg	MD		
Rosen; Craig A.	Laytonsville	MD		

US-CL-CURRENT: 435/69.2; 435/252.3, 435/320.1, 435/325, 435/440, 435/69.1,
435/91.41, 536/23.1, 536/23.5

Full	Title	Citation	Front	Review	Classification	Date	Reference	Abstract	Abstract	Claims	KWIC	Drawn De
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3. Document ID: US 6391853 B1

L6: Entry 3 of 3

File: USPT

May 21, 2002

US-PAT-NO: 6391853

DOCUMENT-IDENTIFIER: US 6391853 B1

TITLE: Human tissue inhibitor of metalloproteinase-4

DATE-ISSUED: May 21, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Greene; John M.	Gaithersburg	MD		
Rosen; Craig A.	Laytonsville	MD		

US-CL-CURRENT: 514/12; 435/226[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Search History](#) | [Edit History](#) | [Claims](#) | [KOMC](#) | [Drawn D](#)[Clear](#) | [Generate Collection](#) | [Print](#) | [Fwd Refs](#) | [Bkwd Refs](#) | [Generate OACS](#)

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Search Results - Record(s) 1 through 10 of 104 returned.

1. Document ID: US 6753176 B2

L4: Entry 1 of 104

File: USPT

Jun 22, 2004

US-PAT-NO: 6753176

DOCUMENT-IDENTIFIER: US 6753176 B2

TITLE: Aggrecan degrading metallo proteases

DATE-ISSUED: June 22, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Arner; Elizabeth C.	West Grove	PA		
Burn; Timothy C.	Hockessin	DE		
Copeland; Robert A.	Hockessin	DE		
Decicco; Carl P.	Newark	DE		
Liu; Ruiqin	Hockessin	DE		
Magolda; Ronald	Wallingford	PA		
Pratta; Michael	Glassboro	NJ		
Solomon; Kimberly A.	Landenburg	PA		
Tortorella; Micky D.	Newark	DE		
Trzaskos; James M.	Boothwyn	PA		
Yang; Fude	Wilmington	DE		

US-CL-CURRENT: 435/226; 435/183, 435/212, 435/219, 435/252.3, 435/320.1, 435/325,
530/350, 536/23.1, 536/23.2, 536/23.5

Full	Title	Citation	Front	Review	Classification	Date	Reference	Abstract	Claims	KMC	Draft	De
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2. Document ID: US 6749853 B1

L4: Entry 2 of 104

File: USPT

Jun 15, 2004

US-PAT-NO: 6749853

DOCUMENT-IDENTIFIER: US 6749853 B1

TITLE: Combined methods and compositions for coagulation and tumor treatment

DATE-ISSUED: June 15, 2004

INVENTOR-INFORMATION:

h e b b g e e e f e f ef b e

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thorpe; Philip E.	Cape Elizabeth	ME		
Edgington; Thomas S.	La Jolla	CA		

US-CL-CURRENT: 424/182.1; 424/178.1, 530/387.1, 530/387.3

[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Sequence](#) | [Anticipated](#) | [Claims](#) | [KMC](#) | [Drawn](#) | [De](#)

3. Document ID: US 6737507 B2

L4: Entry 3 of 104

File: USPT

May 18, 2004

US-PAT-NO: 6737507

DOCUMENT-IDENTIFIER: US 6737507 B2

TITLE: Antiangiogenic peptides and compositions thereof

DATE-ISSUED: May 18, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Moses; Marsha	Brookline	MA		
Wu; Inmin	Boston	MA		
Fernandez; Cecilia	Boston	MA		

US-CL-CURRENT: 530/300; 530/324, 530/325, 530/326, 530/327, 530/328

[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Sequence](#) | [Anticipated](#) | [Claims](#) | [KMC](#) | [Drawn](#) | [De](#)

4. Document ID: US 6706266 B1

L4: Entry 4 of 104

File: USPT

Mar 16, 2004

US-PAT-NO: 6706266

DOCUMENT-IDENTIFIER: US 6706266 B1

TITLE: Oncostatin M antagonists

DATE-ISSUED: March 16, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Life; Paul F.	Stevenage			GB

US-CL-CURRENT: 424/139.1; 424/184.1, 424/278.1

[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Sequence](#) | [Anticipated](#) | [Claims](#) | [KMC](#) | [Drawn](#) | [De](#)

5. Document ID: US RE38386 E

L4: Entry 5 of 104

File: USPT

Jan 13, 2004

US-PAT-NO: RE38386

DOCUMENT-IDENTIFIER: US RE38386 E

TITLE: Retardation of metalloproteinase incidental to HIV and/or AIDS

DATE-ISSUED: January 13, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Berman; Charles L.	New York	NY	10024	

US-CL-CURRENT: 514/152

Full	Title	Citation	Front	Review	Classification	Date	Reference	Abstract	Patent	Claims	RWMD	Drawn De
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 6. Document ID: US 6673333 B1

L4: Entry 6 of 104

File: USPT

Jan 6, 2004

US-PAT-NO: 6673333

DOCUMENT-IDENTIFIER: US 6673333 B1

TITLE: Functional MRI agents for cancer imaging

DATE-ISSUED: January 6, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Meade; Thomas J.	Altadena	CA		
Fraser; Scott	La Canada	CA		
Jacobs; Russell	Arcadia	CA		

US-CL-CURRENT: 424/9.35; 424/9.363

Full	Title	Citation	Front	Review	Classification	Date	Reference	Abstract	Patent	Claims	RWMD	Drawn De
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 7. Document ID: US 6670147 B1

L4: Entry 7 of 104

File: USPT

Dec 30, 2003

US-PAT-NO: 6670147

DOCUMENT-IDENTIFIER: US 6670147 B1

TITLE: Nucleic acid construct for expressing active substances which can be activated by proteases, and preparation and use

DATE-ISSUED: December 30, 2003

h e b b g e e e f e f ef b e

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Heidtmann; Hans Heinrich	Marburg			DE
Mueller; Rolf	Marburg			DE
Sedlacek; Hans-Harald	Marburg			DE

US-CL-CURRENT: 435/69.1; 435/252.3, 435/325, 435/70.1, 530/350, 536/23.1

[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Assignee](#) | [Inventor](#) | [Claims](#) | [KUMC](#) | [Drawn De](#)

8. Document ID: US 6653283 B1

L4: Entry 8 of 104

File: USPT

Nov 25, 2003

US-PAT-NO: 6653283

DOCUMENT-IDENTIFIER: US 6653283 B1

TITLE: Pharmaceutical compositions comprising troponin subunits, fragments and analogs thereof and methods of their use to inhibit angiogenesis

DATE-ISSUED: November 25, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Moses; Marsha A.	Brookline	MA		
Langer; Robert S.	Newton	MA		
Wiederschain; Dimitri G.	Brighton	MA		
Wu; Inmin	Newton	MA		
Sytkowski; Arthur	Arlington	MA		

US-CL-CURRENT: 514/12; 514/2, 514/21, 530/324, 530/350

[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Assignee](#) | [Inventor](#) | [Claims](#) | [KUMC](#) | [Drawn De](#)

9. Document ID: US 6638621 B2

L4: Entry 9 of 104

File: USPT

Oct 28, 2003

US-PAT-NO: 6638621

DOCUMENT-IDENTIFIER: US 6638621 B2

** See image for Certificate of Correction **

TITLE: Coated particles, methods of making and using

DATE-ISSUED: October 28, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Anderson; David	Colonial Heights	VA		

h e b b g e e e f e f ef b e

US-CL-CURRENT: 428/402.24; 424/422, 424/426, 424/450, 435/176[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Search Dates](#) | [Document Types](#) | [Claims](#) | [RIMC](#) | [Draw. De](#)**□ 10. Document ID: US 6635285 B2**

L4: Entry 10 of 104

File: USPT

Oct 21, 2003

US-PAT-NO: 6635285

DOCUMENT-IDENTIFIER: US 6635285 B2

TITLE: Shark cartilage extract: process of making, methods of using, and compositions thereof

DATE-ISSUED: October 21, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Dupont; Eric	Quebec			CA
Brazeau; Paul	Quebec			CA
Juneau; Christina	Quebec			CA
Maes; Daniel H.	Huntington	NY		
Marenus; Kenneth	Dix Hills	NY		
Beliveau; Richard	Quebec			CA

US-CL-CURRENT: 424/548; 424/520, 424/549, 514/859, 514/863, 530/350, 530/400,
530/412, 530/414, 530/415, 530/417, 530/418, 530/422, 530/857[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Search Dates](#) | [Document Types](#) | [Claims](#) | [RIMC](#) | [Draw. De](#)[Clear](#) | [Generate Collection](#) | [Print](#) | [Fwd Refs](#) | [Bkwd Refs](#) | [Generate OACS](#)

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<u>L5</u>	rosen.in.	2229	<u>L5</u>
<u>L4</u>	TIMP-1 and L3	104	<u>L4</u>
<u>L3</u>	l1 and L2	40558	<u>L3</u>
<u>L2</u>	TIMP or tissue inhibitor metalloproteinase	266364	<u>L2</u>
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1. Document ID: US 6749853 B1

L13: Entry 1 of 67

File: USPT

Jun 15, 2004

US-PAT-NO: 6749853

DOCUMENT-IDENTIFIER: US 6749853 B1

TITLE: Combined methods and compositions for coagulation and tumor treatment

DATE-ISSUED: June 15, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thorpe; Philip E.	Cape Elizabeth	ME		
Edgington; Thomas S.	La Jolla	CA		

US-CL-CURRENT: 424/182.1; 424/178.1, 530/387.1, 530/387.3

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2. Document ID: US 6706266 B1

L13: Entry 2 of 67

File: USPT

Mar 16, 2004

US-PAT-NO: 6706266

DOCUMENT-IDENTIFIER: US 6706266 B1

TITLE: Oncostatin M antagonists

DATE-ISSUED: March 16, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Life; Paul F.	Stevenage			GB

US-CL-CURRENT: 424/139.1; 424/184.1, 424/278.1

[Full](#) [Title](#) [Citation](#) [Front](#) [Review](#) [Classification](#) [Date](#) [Reference](#) [Abstract](#) [Detailed Description](#) [Claims](#) [KUDC](#) [Drawn D](#)

3. Document ID: US RE38386 E

h e b

b g e e e f

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ef b e

L13: Entry 3 of 67

File: USPT

Jan 13, 2004

US-PAT-NO: RE38386

DOCUMENT-IDENTIFIER: US RE38386 E

TITLE: Retardation of metalloproteinase incidental to HIV and/or AIDS

DATE-ISSUED: January 13, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Berman; Charles L.	New York	NY	10024	

US-CL-CURRENT: 514/152[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Image](#) | [Attachments](#) | [Claims](#) | [KMMC](#) | [Drawn](#) | [Def](#) 4. Document ID: US 6673333 B1

L13: Entry 4 of 67

File: USPT

Jan 6, 2004

US-PAT-NO: 6673333

DOCUMENT-IDENTIFIER: US 6673333 B1

TITLE: Functional MRI agents for cancer imaging

DATE-ISSUED: January 6, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Meade; Thomas J.	Altadena	CA		
Fraser; Scott	La Canada	CA		
Jacobs; Russell	Arcadia	CA		

US-CL-CURRENT: 424/9.35; 424/9.363[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Image](#) | [Attachments](#) | [Claims](#) | [KMMC](#) | [Drawn](#) | [Def](#) 5. Document ID: US 6638621 B2

L13: Entry 5 of 67

File: USPT

Oct 28, 2003

US-PAT-NO: 6638621

DOCUMENT-IDENTIFIER: US 6638621 B2

** See image for Certificate of Correction **

TITLE: Coated particles, methods of making and using

DATE-ISSUED: October 28, 2003

h e b b g e e e f e f ef b e

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Anderson; David	Colonial Heights	VA		

US-CL-CURRENT: 428/402.24; 424/422, 424/426, 424/450, 435/176

[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Description](#) | [Detailed Description](#) | [Claims](#) | [KMC](#) | [Draw. De](#)

6. Document ID: US 6635285 B2

L13: Entry 6 of 67

File: USPT

Oct 21, 2003

US-PAT-NO: 6635285

DOCUMENT-IDENTIFIER: US 6635285 B2

TITLE: Shark cartilage extract: process of making, methods of using, and compositions thereof

DATE-ISSUED: October 21, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Dupont; Eric	Quebec			CA
Brazeau; Paul	Quebec			CA
Juneau; Christina	Quebec			CA
Maes; Daniel H.	Huntington	NY		
Marenus; Kenneth	Dix Hills	NY		
Beliveau; Richard	Quebec			CA

US-CL-CURRENT: 424/548; 424/520, 424/549, 514/859, 514/863, 530/350, 530/400,
530/412, 530/414, 530/415, 530/417, 530/418, 530/422, 530/857

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7. Document ID: US 6627605 B1

L13: Entry 7 of 67

File: USPT

Sep 30, 2003

US-PAT-NO: 6627605

DOCUMENT-IDENTIFIER: US 6627605 B1

TITLE: Human proteinase molecules

DATE-ISSUED: September 30, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bandman; Olga	Mountain View	CA		
Corley; Neil C.	Mountain View	CA		
Guegler; Karl J.	Menlo Park	CA		

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Baughn; Mariah R. San Jose CA

US-CL-CURRENT: 514/12; 435/7.1, 530/350

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8. Document ID: US 6620606 B2

L13: Entry 8 of 67

File: USPT

Sep 16, 2003

US-PAT-NO: 6620606

DOCUMENT-IDENTIFIER: US 6620606 B2

TITLE: Human cathepsin

DATE-ISSUED: September 16, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bandman; Olga	Mountain View	CA		
Guegler; Karl J.	Menlo Park	CA		
Corley; Neil C.	Mountain View	CA		
Shah; Purvi	Sunnyvale	CA		

US-CL-CURRENT: 435/219; 435/226, 435/252.33, 435/69.1, 536/23.2, 536/23.5, 536/24.1

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9. Document ID: US 6613322 B2

L13: Entry 9 of 67

File: USPT

Sep 2, 2003

US-PAT-NO: 6613322

DOCUMENT-IDENTIFIER: US 6613322 B2

TITLE: Method for treating a subject suffering from conditions associated with an extracellular zinc sphingomyelinase

DATE-ISSUED: September 2, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Tabas; Ira	New York	NY		
Schissel; Scott L.	Westwood	MA		
Williams; Kevin Jon	Wynnewood	PA		

US-CL-CURRENT: 424/94.6; 424/94.1, 424/94.3

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10. Document ID: US 6586401 B1

L13: Entry 10 of 67

File: USPT

Jul 1, 2003

US-PAT-NO: 6586401

DOCUMENT-IDENTIFIER: US 6586401 B1

TITLE: Troponin subunit I fragment and homologs thereof

DATE-ISSUED: July 1, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thorn; Richard M.	North Easton	MA		
Lanser; Marc E.	Dover	MA		
Moses; Marsha A.	Brookline	MA		
Wiederschain; Dmitri G.	Dighton	MA		

US-CL-CURRENT: 514/13; 530/326

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